The Diagnosis of Blindness

JACKSON, EDWARD

HV1594
J

M.C. MIGEL LIBRARY
AMERICAN PRINTING
HOUSE FOR THE BLIND

HV1594
J
copy 1

36422-MIGEL

THE DIAGNOSIS OF BLINDNESS *

EDWARD JACKSON, M.D.
DENVER

A diagnosis is a decision, to be reached by careful consideration of all obtainable evidence. In a military commander or an executive, indecision may be more disastrous than a wrong decision. For a physician or surgeon, it may be quite as serious in its consequences. In cases of blindness, a decision on the extent or the cause of blindness may be the only professional service we can render. Every attempt to assist, to train, to compensate or to make economic provision for the blind renders the diagnosis of blindness more important.

All human decisions are liable to error, but this does not make decision less important. Through ignorance of facts, distortion of them, prejudice, poor judgment, failure properly to appraise the relative value of evidence, wrong conclusions may be reached. This may be the case in military affairs or in executive business, with judges as to points of law, or juries in regard to matters of fact. But this does not relieve officers or executives, judges or juries from doing their best to reach a decision on the situation or the facts presented. It is equally the duty of the physician to reach a diagnosis, and especially of the ophthalmologist to reach a decision—a diagnosis—regarding blindness.

If he is too anxious to avoid criticism for apparent dogmatism, to make clear what he really believes is the truth, his statement may be of no service to truth, justice or science. It is enough in any case to preface his conclusions by the simple words, "I think," or "It is my conclusion," "I believe that this person or this eye is, or is not, blind; or that the blindness is, or is not, from this or that particular cause." To decline to reach and state a definite conclusion as to the existence, degree and etiology of blindness is, in most cases, equivalent to a confession of incompetence, laziness, or indifference to the question presented.

No opinion should be rendered regarding a case of blindness without seeking out and considering all the evidence bearing on it. But, having done this, it is a shirking of responsibility for any one holding himself before the community as an expert as to diseases and defects of the eye and vision, to refuse to give a diagnosis as to blindness, or to state it in such doubtful terms as to render that diagnosis worthless.

It may be felt that, vision being subjective, no one but the individual himself can tell whether or not he is blind. It is equally true that the individual must generally know better than any one else whether he has committed a murder or theft, or planned and assisted in any crime, or really is justly entitled to some piece of property he has claimed. But as in any of those cases, the interest of the claimant in his alleged blindness often renders his testimony worthless. If his testimony is admitted, it gains significance and weight only as it is corroborated by collateral evidence over which the claimant's self interest has no vitiating influence. To elicit, weigh and present such unbiased evidence, and to estimate its value; in other words, to bring out and estimate the value of objective evidence of blindness is the business of the ophthalmologist.

The diagnosis should answer these questions:

Is there blindness present?
How great is the impairment of sight?
What is the cause of it?

With what certainty and accuracy can these be answered by a careful and complete objective examination? Let us admit that the diagnosis of blindness is easiest, most certain and most exact when the cooperation of the patient brings to our assistance all the subjective evidence regarding vision. But when the patient or claimant will not so cooperate, will not give such assistance, and by refusal to work actively against what he conceives to be his interest, either by a general attitude of obstruction, or direct falsehood, would prevent a correct or accurate diagnosis, what may we do to bring out the facts by complete and conscientious objective examination?

The answer to this question determines our ability to assist the community in excluding from certain occupations or branches of service those whose defective sight would endanger themselves, or impair their efficiency in those lines of service. It becomes more important with all legislation bearing on industrial insurance, compensation for injury, assistance, special privileges or benefit for blindness—directions in which the recognition of social responsibility has developed very rapidly of late. If we cannot render the objective diagnosis of blindness reliable, within practical limits, such legislation, custom or sentiment as has shown this development merely places the honest, industrious, kindly part of the community at the mercy of the morbid whims, cupidity, falsehood and selfishness of those whose extinction would benefit the race. Progress in the direction of benevolence, charity, public security or justice with regard to blindness rests today on its objective diagnosis.

SOURCES OF EVIDENCE

The history of the individual case, even the history of subjective phenomena, may have all the significance and value of objective evidence. Thus, a history of recent sudden blindness, perhaps following accident, when examination shows old scars of the cornea or choroid, or vitreous opacity, either congenital or manifestly of long standing, is absolute evidence of falsehood, either intentional or from ignorance, that properly discredits all evidence from such a source. On the other hand, the history of sudden blindness in homonymous lateral half fields, supported by corroborative limitations of the fields, and other evidences of intracranial lesions, becomes highly valuable and significant. Every cause of blindness that we know links up with an appropriate and suggestive case history. Allowing for the fact that no two cases are exactly alike in all particulars, it is next to impossible for any patient or claimant to construct a false history of his troubles that will pass critical consideration from an expert, unless he has the assistance of expert knowledge and experience that rarely lends itself to the purposes of the criminal. The case history carefully taken and considered, repeated if necessary and made the basis of cross examination, will, in the great majority of cases, reveal how much or how little credence can be given to the statement of the patient or claimant, and suggest in what directions objective examinations should be pushed to discover the truth.

The external examination of the eye in all cases of alleged blindness, complete or partial, should be carefully planned, prolonged and thorough. Every objec-

---

* Read before the Section on Ophthalmology at the Seventy-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., May, 1925.

* Because of lack of space, this article is abbreviated in The Journal. The complete article appears in the Transactions of the Section and in the author's reprints.

ABSTRACT OF DISCUSSION

Dr. F. M. Johns, New Orleans: Any one who has followed the history of plague will be reminded that in a seaport city it is simply a question of time, it may be ten or fifty years, when it will break out; and it is wise to keep in mind the progress of measures directed against the disease. The only question that probably requires some comment in Dr. Lloyd's paper is the treatment of plague with serum. I think I can agree with him that in the review of the literature the only treatment of bubonic plague is with the antiplague serum. The result of serum in cases that are diagnosed quickly is probably just as spectacular as with any other serum treatment. I see that Dr. Seemann is present and would like to have him discuss the cases he treated in New Orleans. We had several patients with temperatures of 105 or 106 F. and almost moribund; serum was administered and in a few hours the temperature dropped and the patients made uneventful recoveries. Naturally, the infection varies. Some epidemics seem to have a more virulent type of organism, but I am satisfied in my own mind that the majority of cases, if treated promptly with fresh serum, would be greatly benefited. The obtaining of serum is also a matter of great importance. The epidemic usually breaks out in a community with probably no provisions having been made for the obtaining of serum. The variety administered is probably several years old. I should like to ask Dr. Lloyd his opinion of the various forms of serum used, also the keeping qualities of serum; in other words, some serums disintegrate in a few months under adverse conditions and I would ask whether this is not responsible for most of the lack of effect reported from the use of serum.

Dr. W. H. Seemann, New Orleans: I am delighted to hear an authority with the large experience of Dr. Lloyd come to the defense of serum in plague. Average textbooks refer to plague treatment with serum almost with derision, and yet I know of no agent more unjustly maligned than antiplague serum. We have had a good deal of experience with bubonic plague in New Orleans. We at first got serum from the Pasteur Institute. It was old, as Dr. Johns referred to in a general way, and it was not made with the specific strain of antigen. In other words, it did not have a specificity against our type of organisms. We subsequently injected two horses ourselves. We used the actual bacteria from patients and rats right in the city, and the use of this serum when finished gave results strikingly different from that we have obtained before, proving that if we want to get the maximum results of serum we must try to get the local specific antigen, inoculate a horse, and produce specific antibodies. I believe I have given the largest doses of serum of any man who has treated plague. I never gave less than 150 or 160 c.c., and I go further than Dr. Lloyd and repeat in from six to eight hours with similar doses. I had one man given 160 c.c. intravenously, and the temperature went from 106.5 F. to normal next morning. It is my belief that there is no difference between pneumonic plague and bubonic plague so far as the causative agent is concerned. I have seen two cases of plague in which there was severe pneumonia, and recently I saw a case of bronchopneumonia. The sputum was literally teeming with plague bacilli. In each one we were able to get results.

Dr. Matthias Nicoll, Jr., Albany, N. Y.: I know very little about plague; but a few days ago Dr. Wu, in charge of the federal control of plague in Manchuria as a representative of the Chinese government, expressed the opinion that the use of the serum was in his experience of very doubtful value.

Dr. Samuel B. Grubbs, New York: Plague is our most important problem, not only because the disease is advancing but because present methods to exclude it are so expensive. We are therefore trying to improve these methods and need your advice and help. We are working on two distinct lines, one the practical which is largely rat catching; and the other the investigation of plague epidemiology, especially the unknown details of its transmission by rats and fleas. We find interesting questions that we hope some day to answer; for example, why the North Atlantic coast has never been infected, although ships come there from all over the world and it should naturally have been infected long ago. Plague has infected several United States ports and exists in three of them at present, but North Atlantic ports have remained free. We cannot give the entire credit to superior quarantine; it is more probable that certain territory is noninfectable or nearly so. We want to learn what makes a port infectable. It may be possible to establish a rat or rat-flea index similar to the yellow fever *Stegomyia* index that will allow us to classify ports according to their infectability and to base our quarantine on this classification. Fumigation of vessels is now our chief defense, and the requirements are the same for all ports regardless of their degree of infectability. In New York we have constantly tried to check our fumigation by trapping and observation. We have learned that the most painstaking fumigation rarely fails to leave behind a few rats, sometimes a considerable number. With favorable conditions this means an increase of the original number within a few months. We have therefore tried to rat-proof ships, which means the elimination or blocking off of all places where rats can hide, the protection from rats of food supplies, and the blocking of rat runs. We are rat-proofing several ships at present and find that the rat population decreases as the rat-proofing progresses.

Dr. E. R. Hayhurst, Columbus, Ohio: I am interested in the possible historical relationship of plagues. In Ohio there was probably as great development of the mound builders as any place in the United States. It has always been a mystery why this people disappeared so suddenly Sudden disappearance of peoples indicates superficial burial if any burial at all. The thought occurred to some of us who are members of the Ohio Archeological and Historical Association that the reason might possibly have been the great plague epidemic of the thirteenth century (1345-1350), as recorded in Europe, because there is good evidence, from mounds of these people, that they were living well within the last thousand years.

Dr. B. J. Lloyd, Washington, D. C.: Answering Dr Grubbs, I may say that the subject of rat-proofing vessels is treated of in the part of the paper which I did not read. To Dr. Nichols I may say that I did not know that Dr. Wu was as pessimistic about the serum treatment of plague as his latest statement would indicate. I wonder whether he had in mind pneumonic plague rather than bubonic. I know that there are outstanding men who do not regard the use of antiplague serum very highly even in the bubonic form of this disease, but I cannot concur in that opinion. I do not know how long antiplague serum will keep at low temperatures. I wrote to a friend of mine, who is working in the Pasteur Institute, but he states that they do not have the information. During the work in Guayaquil, I received a consignment of antiplague serum from Dr. Perry, who was then stationed on the Isthmus of Panama. It had been kept six months; under what conditions, I do not know. Its appearance made me think that it was of questionable value, but, as serum was scarce, I decided to use it. After losing several patients who I thought should have recovered, I discontinued its use and sent the remainder back to Dr. Perry The serum used at Guayaquil, Ecuador, I purchased from the Pasteur Institute, Paris, from an institution in Berne, Switzerland, and from another in Buenos Aires. Purchases of from 10,000 to 20,000 20 c.c. bottles were made frequently, the serum being requested by cable and arriving fresh. We could tell from the markings when the serum was drawn. I have found difficulty in obtaining serum in the United States that was bright and clear, and I do not know of any serum manufactured in the United States since 1923. When buying serum, I think it is well to insist that the dealer state when the serum was drawn. I prefer serum as fresh as can be had. I think it is quite possible, as suggested by Dr. Johns and Dr. Seemann, that a more potent serum can be made from the particular strain of plague present in a given vicinity, although serum when so made is of course used fresh, which is a decided advantage. With regard to the identity of the organism which causes bubonic plague and that which causes pneumonic plague, I think there can be no reasonable question.

tive sign of previous disease should be noted and carefully considered with reference to its developmental or pathologic possibilities, the suggestions it makes of other lines of investigation, and the necessary or possible influence it may exert on visual acuity. A scar not perceptible to casual inspection may greatly impair visual acuity by the astigmatism, regular or irregular, that it causes. Only by eliminating or properly estimating all other factors causing impairment of vision can the right understanding of visual impairment by any one condition be determined. The objective diagnosis of blindness is the summing up of all ophthalmic diagnosis, in which common and generally unimportant conditions cannot be neglected without introducing sources of serious error and uncertainty.

The ophthalmoscopic examination of the eye contributes equally to our knowledge of pathologic conditions present, and in addition it furnishes a test of the influence such conditions exert to impair sight in the particular eye under examination, by impairing the transmission of light through the refractive media and surfaces of the eye. The light coming out of the eye from behind the retina must pass through exactly the same physical conditions, to reach the eye of the observer, as light encounters after entering the eye from the external world, before it reaches the percipient elements of the retina of the patient. By familiarity with the details of the ocular fundus and the fundus reflex when fundus details are not visible, we secure at the back of the eye a series of test objects, the visibility of which can be compared with the visibility of the test letters and figures we use in everyday tests for visual acuity. By comparing visual acuity, tested in the ordinary way, in patients giving full cooperation in such tests, with the ophthalmoscopic visibility of the fundus, we can construct an approximate scale of fundus visibility that will correspond roughly to the visual acuity scale that we work with in general ocular examinations. This scale can be applied largely without the patient's cooperation, being in effect an objective method of examination.

In ophthalmoscopic estimation of visual impairment, skiascopy, from near the point of reversal, gives important evidence as to complete transparence of the ocular media and the share that ametropia, especially irregular astigmia, may have in producing visual impairment. Two eyes may appear to present about the same amount of opacity in the dioptric media; but in one skiascopy will be found impossible and in the other easy. It is safe to infer from the test that in the first visual acuity is greatly affected by the opacity, and that in the other it is not.

Each of us has developed some such scale for judging the effect of lesions impairing the transmission of light through the dioptric media, on which he bases his judgment as to the amount of visual impairment to be expected by a certain haziness of the fundus details. The noting as exactly as possible such interference with seeing the disk, central vessels, primary and secondary branches and smaller arterial and venous twigs, pigment stippling, reflexes, etc., and comparing such notes with those of visual acuity in cooperating patients, will give each observer a better grasp of ophthalmoscopic diagnosis and will indicate just what details should be looked for and what significance can be attached to their observed visibility. From the series of cases given below of opacity presented in the cornea, lens or vitreous, in each an attempt has been made to construct the outline of a scale of objective evidence of visual impairment. If a number of ophthalmologists will gather and compare such data, it will add to the value of what each of us has already learned in this direction, by his own examinations of patients.

The method pursued for estimating the effects of opacity and irregular refraction in the refracting media may be modified for application to all observed deviations of the ocular tissues from their normals. We can record the inflammatory and degenerative changes in the retina, choroid and optic disk with the visual impairment associated in each case in which the patient cooperates in giving his exact vision; and from such records we might construct a practical scale of probable impairments of vision, for the different visible ophthalmoscopic changes that may be observed.

Along these lines we must seek answers to such questions as: What visual disturbance can be expected with simple detachment of the retina? What from a well marked glaucoma cup? How much importance can be attached to whiteness of the optic disk, as evidence of visual impairment by optic atrophy? To what extent do pigment migrations imply destruction of visual function?

When it comes to assessing the visual damage due to fundus lesions, a damage arising from structural and functional impairment of the tissues inspected, we are on more uncertain ground. But when these furnish the best evidence obtainable, apart from the claimant's statements of what he sees, every effort should be made to ascertain the probable significance of what we can see.

Certain definite and measurable lesions, such as detachment of the retina, have a positive, although not entirely exact influence, on visual acuity. Series of two of these, detachment of the retina and the cupping of the optic disk in chronic glaucoma, are included in cases grouped below for study. Certain less conspicuous changes in the macula, likely to exist with circinate retinitis, are also important evidences of impaired vision.

In the appended series of retinal and choroidal lesions, the records regarding macular conditions have been emphasized by always including them in the case abstracts, which seem to show their importance. But wide acquaintance with anomalous appearances of the macula compatible with normal vision is essential to the estimation of the visual significance of what is there seen.

Exact observations as to color are of great importance for the estimation of visual capacity in the retina and optic nerve. The cases of optic atrophy are interesting as regards color significance. In this connection, careful observation of the finer or nutrient vessels is of paramount importance, as in the deductions to be made from swelling of the optic nerve head. This is possible to us as regards the optic nerve, with carefully chosen light for ophthalmoscopy, and is probably equally available for the retina, when red-free light is systematically applied for that purpose. But it must be borne in mind that the exact color of the light used has enormous importance as influencing the appearances to be observed in all such examinations. The colors employed must be standardized before the significance of what is noted can be properly estimated.

The application of observations made with the corneal microscope promises a great deal of help in the diagnosis of blindness in the future. But a great many more of these observations must be made and carefully compared with corrected visual acuity before due importance can be attached to such findings. An instance of possible advances in this direction is the case of tuberculous uveitis cited in connection with corneal opacities (Case 22).

The testing of pupil reflexes and ocular movements to avoid diplopia may give extremely important evidence as to blindness that may be present. It is fairly established that the direct pupil reflex to light implies a reflex arc, stretching from the retina to the primary visual centers along the afferent tracts, sufficiently effective to perform its function in a way that disproves complete blindness. Even a moderate direct pupil reflex is evidence of some vision, unless the visual tracts are interrupted beyond the primary centers by lesions affecting the optic radiations or visual cortex.

The evidence obtained by the direct light reflex is confirmed by the consensual light reflex in all cases of claimed monocular blindness. If the afferent tract is interrupted in front of the primary visual centers, the consensual light reflex is also lost for light thrown on the blind eye. But for loss of function behind the primary centers, we have homonymous hemianopia and the hemianopic pupil inaction; so that the hemianopic pupil reaction has great practical importance in the diagnosis of blindness, as well as in the localization of lesions affecting the visual tract.

The attempts made by Hess and others to study the hemianopic pupil reaction by light impressions sharply localized on small parts of the retina within the so-called pupillomotor area have led to confusion and doubt as to the diagnostic value of this reaction. The method described by Fisher,[1] if carefully followed, seems, however, to give definite and positive clinical data. If, with positive direct or consensual clinical reaction of the pupil to light, such reaction is found to be about the same whether the light falls chiefly on the right or on the left half of the retina, we are justified in concluding that the visual pathways are not interrupted behind the primary centers—that the person giving such reactions has light perception, and that if the reactions are good he has good light perception.

The consensual reaction is of especial importance when disease in one eye has bound down the pupil; and, it is asserted, has also caused blindness in that eye, the iris of the other remaining free to react under the influence of light. It is in connection with the pupil reactions that the claimant's statements with regard to the circumstances and nature of the disability claimed become of great importance. In the cases cited below, the condition of the pupil reflex has often not been noted on the case records, and the data here quoted are confined strictly to those found in the record. In general, no record regarding the pupil might be taken to mean that the reflexes were normal. But this is not satisfactory evidence when the pupil reflex may be the deciding factor as to whether blindness claimed is or is not really present.

It must be understood that in the foregoing discussion the ordinary reflexes of the pupil to light are alone referred to—those that take place within a very few seconds after exposure to the light stimulus. There is also a slow reaction of the pupil to strong light, and especially to direct sunlight, to which attention was called by the late George C. Harlan,[2] in which the pupil contracts slowly for several minutes. This form of reaction is not well understood and apparently has not been generally studied. It seems not dependent on the generally recognized pupillary reflex arc.

OCULAR MOVEMENTS TO AVOID DIPLOPIA

If vision is good in both eyes, and the faculty of binocular fusion is well developed, certain movements of the eye to avoid diplopia when a prism is placed before it, or withdrawn, are quite involuntary, and their absence may be taken as good evidence that binocular vision is not present. On the other hand, when these movements are elicited by placing a prism before one eye and removing it, we have proof positive that binocular vision is present.

Of course, a patient may have good vision in each eye and yet have no power of binocular vision or to perceive binocular diplopia, and such a person will give no ocular movements in response to such use of prisms; but instances of such lack of binocular vision and diplopia are relatively rare, and detract little from the value of this test in the diagnosis of blindness. If the antidiplopic movements of the eye are elicited by this use of prisms, especially on repeated trials, it is proof positive that blindness of either eye is not present.

It is quite possible by careful selection of test objects to be presented to the eyes, and the background against which they are to be seen, to form with this test an approximate estimate of the degree of visual acuity present. But methods of making this a quantitative visual test should be worked out by careful experiment.

CASES OF BLINDNESS

The following cases are cited by giving the vision present, with a few of the leading objective conditions noted in each, such as are of most value for the objective diagnosis of blindness. The vision is given in the terms originally used in the case records, although in computing averages, as for lens opacities of different degrees, the expressions noted have been reduced to equivalent decimals, for convenience in addition and division. They are the methods of noting visual acuity in common use.

The designation by distance at which fingers can be counted ought to be discarded; and of late years I have discarded it. It depends largely on the background against which the fingers are held. The regular visual tests, the broken ring, the incomplete square, the cross-point test of Wolfberg, or Ewing's broken line test can be used for the degrees of visual impairment commonly tested by counting fingers; and they give more accurate results. Roughly speaking, counting fingers is about equal to, or a little better than, recognition of 200 foot letters or tests, at an equal distance.

Only such cases are cited here as have been examined in private practice, in which the patients have given full cooperation to bring out their best vision, and have been under observation long enough to eliminate chances of error from a single examination. The facts cited are often those observed at one time in the prolonged course of a case in which many changes in vision and objective symptoms were observed. They have all been cases in which vision was reduced to about one tenth or less, cases approaching or attaining the condition of "industrial blindness."

---

[1] Fisher, J. H.: Tr. Ophth. Soc. U. K. **41**:7.

[2] Harlan, G. C.: Tr. Am. Ophth. Soc. **7**: 671, 1894-1896.

OPACITIES OF THE MEDIA

This is the class of cases in which the visual impairment is presumably due to interference with the transmission of light to the percipient layer of the retina; and so far as possible cases in which blindness or impairment of vision was largely due to other causes have been excluded. In these cases the transmission of light from the ocular fundus to the observer's eye furnishes an objective scale, with which the transmission of light from without inward may be deduced with probability and some exactness.

These cases are here separated into three groups; opacities of the crystalline lens, of the cornea and of the vitreous.

LENS OPACITIES

Of opacities of the lens, 100 cases were taken, including 113 eyes. When the visual acuity is reduced in such cases to one tenth or less, the opacity generally affects the whole area of the pupil for light that reaches the macula through it from any external object. In nuclear cataract the periphery of the retina may receive light coming obliquely through relatively clear cortex, but this does not greatly affect the visual acuity as usually measured at the macula. In many of the cases the visual acuity was known before the lens opacity developed, and in some the vision afterward obtained by removal of the cataract is given. The visual impairment is thus definitely correlated with the observed objective symptoms.

Of these 113 eyes, eight are recorded as having no light perception. Presumably conditions other than lens opacity had arisen after cataract formation in these eyes to render the blindness absolute. But where no indication could be found as to what such condition might be, the case was included for comparison with cases of less extreme blindness, and of similar blindness from other causes. Also because the patient's response of "no perception of light" depends a good deal on his mentality. We constantly meet patients who claim to perceive light, although the most careful testing brings out no evidence to support their claim; and, on the other hand, there are cases recorded as having "no light perception" in which later removal of extensive vitreous hemorrhage, or a black cataract, has been followed by return of quite useful vision.

Patient 14 had light perception but nothing more. These represent the class of mature dense cataracts. Patient 23 could recognize "moving shadows"; that is, they could tell in what direction the shadows moved across the field of vision.

Twenty were able to "count fingers" at distances ranging from 3 inches to 4 feet, the average being 15½ inches; ten had vision ranging from 1/500 to 1/60, averaging 0.01; nineteen had vision ranging from 1/50 to 1/25, averaging 0.031; twelve had vision ranging from 1/20 to 7/100, averaging 0.058; nine had vision ranging from 8/100 to 10/100, averaging 0.085.

CORNEAL OPACITIES

Of the corneal opacities there were seventy cases, eighty eyes. Among these five had no light perception, which had been lost by the disease or injury causing the corneal opacity; nineteen had only light perception; seven could "see moving shadows"; nine could count fingers at distances varying from 6 inches to 4 feet, and averaging $15\frac{3}{10}$ inches; twelve had vision ranging from 1/500 to 1/60, averaging 0.009; thirteen had vision ranging from 1/50 to 1/25, averaging 0.026; eight had vision ranging from 1/20 to 1/15, averaging 0.052; seven had vision ranging from 8/100 to 10/100, averaging 0.088.

VITREOUS OPACITIES

There were forty-one cases of vitreous opacities, forty-four eyes. Of these, three had no light perception; four had only light perception; eight could see moving shadows; seven could count fingers at from 7 to 18 inches, averaging $10\frac{2}{10}$ inches; four had vision of from 1/500 to 1/60, averaging 0.009; nine had vision of from 1/50 to 1/22, with an average of 0.034; two had vision of from 1/20 to 1/15, averaging 0.058; and five had vision of from 8/100 to 13/100, averaging 0.1.

RELATION OF VISION TO VISIBILITY OF THE FUNDUS

In these 211 cases of opacity of the media we find 205 eyes with regard to which the veiling of the fundus is recorded in definite terms that can be arranged under the following heads: no fundus reflex; moderate fundus reflex; good fundus reflex; details of the fundus dimly seen and details well seen. As to the visual acuity that these grades of visibility of the fundus may imply, we gather the following statistics:

No fundus reflex was visible in ninety-one eyes. Of these, twelve had no perception of light, thirty-three had only light perception, thirty could see moving shadows, ten could count fingers, and six had vision recorded as ranging from 0.001 to 0.04 and averaging 0.015.

There was a moderate fundus reflex in fifty-one eyes. Of these, ten had only light perception, four saw moving shadows, thirteen could count fingers, and twenty-four had vision of from 1/500 to 8/100.

There was a good fundus reflex, but no fundus details were visible, in twenty-nine eyes. Of these, six could only count fingers, while in twenty-three the vision ranged from 1/200 to 8/100.

Details of the fundus were dimly seen in thirty-four eyes. Of these, four could see only moving shadows and five could count fingers, while in twenty-five vision ranged from 3/500 to 1/10.

Details of the fundus were clearly seen in three cases. All of these patients had vision of from 4/100 to 10/100.

FUNDUS LESIONS

In general, it is scarcely possible to form a scale of the influence of visible lesions of the ocular fundus on the acuity of vision. However, there are three conditions in which the ophthalmoscopic appearances give some indications for quantitative grading of the impairment of vision they cause. These are: detachment of the retina, glaucoma, and atrophy of the optic nerve.

DETACHMENT OF RETINA

Among thirty-three cases of detached retina, we find but one eye that had no light perception, and in this case the detachment was traumatic. There were four eyes in which vision was reduced to light perception, and twelve in which it was only moving shadows. In seven eyes, it was recorded as counting fingers at from 6 inches to 4 feet, and in nine it varied from 1/100 to 4/60.

GLAUCOMA

Of forty-three cases of glaucoma, fifty-four eyes, there were twenty-two cases in which the media were so hazy that the fundus could not be seen, but in all the others, twenty-one eyes, glaucoma cupping was present.

In sixteen cases there was no light perception. In five ceses, six eyes, vision was reduced to light perception.

In ten cases, twelve eyes, moving shadows were perceived in some part of the field. In three cases, three eyes, fingers were counted at from 6 inches to 2 feet.

In nine cases, thirteen eyes, vision of from 1/100 to 1/10 remained in some part of the visual field.

OPTIC NERVE ATROPHY

Of atrophy of the optic nerve, there were thirty-two cases, forty-one eyes. Among these, in thirteen eyes of eleven patients there was no perception of light. In three eyes of three patients, there was only light perception. One eye saw only moving shadows. Four eyes in as many patients could count fingers. In the twenty eyes remaining, vision ranged from 1/125 to 9/100. In four of these eyes with some vision, there were still some swelling, haze and vascularity of remaining neuritis. In all the others, the nutrient vessels of the disk were almost or quite lacking, and gray or "paper white."

RETINAL DISEASE

In forty-three cases of retinal disease other than retinal detachment, revealed by ophthalmoscopy, the lesions vary so widely that no general grouping of similar conditions would be profitable, although the study of individual conditions is instructive. It should be noted, however, that in only three of the eyes was light perception lacking, and in all of these there was general disease of the retina, one of general degeneration, probably cystic, one of atrophy and one of neuro-epithelioma. Of the thirty-eight eyes in which vision ranged from 1/240 to 1/10, lesions of the macula are noted in fifteen. Any examination for causes and degree of blindness ought to include careful ophthalmoscopic examination of the macula.

CHOROIDAL DISEASE, DEGENERATION AND ATROPHY

In the fifty-one cases, fifty-nine eyes, given above as suffering chiefly from choroidal disease, the involvement of the macula is still more important. In twenty-three cases, twenty-five eyes, the involvement of the macular region of the choroid is especially noted; in the three eyes that had no light perception, coloboma of the choroid (probably with some other malformation). Sarcoma and universal sclerosis of the choroidal vessels following injury are mentioned.

PANOPHTHALMITIS AND SHRUNKEN GLOBE

In panophthalmitis in eleven eyes, the best vision was counting fingers at 30 inches, and six eyes had no light perception.

RETROBULBAR NEURITIS; TOBACCO AMBLYOPIA

In nerve lesions, retrobulbar neuritis and tobacco amblyopia, the poorest vision was perception of moving shadows; and this was an acute condition, followed by recovery of vision 2/3 and 9/10.

VISUAL TRACTS AND CENTERS

Among the four cases, eight eyes, in disorders of the visual tracts and centers, the best vision was 2/100 in one patient. Two saw moving shadows and one had no light perception.

AMBLYOPIA, CONGENITAL OR WITH SQUINT

It is understood that in these forty-seven cases two distinct conditions, congenital amblyopia, and amblyopia arising in connection with strabismus, are classed together; but in the absence of explanatory ocular lesions, they are alike so far as concerns objective symptoms. The diagnosis of them, including the discrimination between them, must rest largely on the history, on the point as to whether the amblyopia is bilateral or unilateral, and on the presence or absence of other congenital deviations from the normal type. It will be noted that of the forty-seven cases, twenty-two showed organic deviations from the normal, which took them out of the class of strict amblyopias. But such organic defects were not sufficient to account for the impairment of vision present.

It will be remembered that in Fuchs' work on the causes and prevention of blindness, congenital amblyopia, given as the cause of 0.2 per cent. of the cases in the statistics of Magnus, is the only cause that did not present characteristic objective evidence on which a diagnosis could be based. In this one condition the diagnosis must be reached through the history, and the absence of objective symptoms. The tests for malingering may be used to prove that the amblyopia is genuine and not merely psychic. But such tests must be multiplied and applied with great care, to furnish a sufficient basis for the diagnosis.

CONCLUSION

From this survey of 704 eyes blind, or near blind, from all the common causes of blindness, this fact stands out: Except in amblyopia, congenital or associated with squint, blindness is always associated with objective changes that declare the sight has been greatly impaired, and often furnish the most important evidence of how great that impairment is.

ABSTRACT OF DISCUSSION

Dr. T. B. Schneideman, Philadelphia: In the scientific sense, an eye is blind when it is incapable of perceiving the sensation of light—amaurosis. Practically, a person is blind when his visual power is so far reduced that he is incapable of following any occupation to gain his livelihood; from this point of view, it is of minor importance whether such a person possesses or not some slight remains of sight. This is the same as the definition of Cohn, one of the early students of blindness. Emmert defines blindness as the impossibility of going about in daylight, thus excluding hemeralopes. Schmidt-Rimpler and Magnus would express the limit of blindness when the patient is just able to count fingers at one-third meter in ordinary daylight. Statistics as to the incidence and causes of blindness among the population of a country depend mostly on official government census, and these are far from exact even in the most civilized countries. The statistics of blindness, as determined by census, give for the United States in 1920 the total number of the blind as 52,567, being 497 per million of population.

The chief causes of blindness given are cataract, glaucoma, atrophy of the optic nerve, ophthalmia neonatorum and trachoma. Only a very small percentage of those born blind reported blindness in the parents. Magnus' statistics, now somewhat antiquated, but still ranking among the most accurate at hand, based on 2,528 cases of blindness, give the leading causes in their relative proportion of frequency as ophthalmia neonatorum, trachoma and conjunctivitis of adults, diseases of the cornea, iridochoroiditis and iridocyclitis, glaucoma, atrophy of the optic nerve, injuries, and detachment of the retina. The question of the existence of blindness of one or both eyes resolves itself in practice to a determination whether the person under examination is simulating or not, or whether he is hysterical; for if the subject is honestly attempting to reply truthfully, the matter is easily settled. The ordinary tests for simulation or self deception are so well known that it is not necessary to enter into details.

The test devised by Dr. Jackson with the crossed cylinders is also based on impairing the vision of the good eye. Its special value as a subjective test is that so little manipulation is required by the examiner, who simply turns one of the pair of cylinders which have been placed in front of the good eye with parallel axes before the examination is begun, and which in that position exert no influence on the vision; the person under examination can scarcely realize how the eye alleged to be poor is being tested. Based as it is on the impairment of vision that results when a convex and a concave cylinder are brought to a right angle, reducing the vision to about 0.4 in an emmetropic eye with + 1 and — 1 cylinders crossed, it furnishes a ready means of disclosing unreliable statements as to the degree of impairment of the eye claimed to be defective.

Dr. Lucien Howe, Buffalo: The question of blindness has always been a *bête noire*. Every asylum for the blind is certainly a standing monument to our ignorance. But as we learn, their number will decrease. I think it is Magnus who says that with the introduction of vaccination blindness in Europe fell something like 40 per cent. We have not been able to make any such change since then, but gradually we are approaching it and little by little we make steps forward. One method for detecting blindness has served me well: A man was sent to me with a partially opaque cornea on both sides, but not enough to account for the almost total blindness that he claimed. I said at once that it was impossible to arrive at a conclusion as to the degree of blindness, and the only thing to do was to send him to the hospital and keep him under observation, which was done. The orderly who had always been connected with the institution was an exceedingly intelligent man; he watched him and I watched him. I was sorry for the poor fellow and I came to the conclusion that his sight was very slight. It seemed to us best, however, to test him still further, so I told the orderly to see how much he was able to get about in the dark and with a diminished light. He could hardly find his way about, it was necessary for the orderly to lead him to different parts of the ward. One evening I suggested to the orderly that he put around this patient's bed a table and a chair and everything that might obstruct his progress. Then I gave the man an active cathartic. At the proper time he arose, rubbed his eyes, and without any trouble found his way around the tables and chairs, leaving no doubt as to the question of diagnosis.

Dr. F. H. Verhoeff, Boston: This paper is very helpful to all of us who have to testify before industrial accident boards. I have used a simple test for many years with great success. Whenever a patient comes in who is suspected of malingering, the first thing I do is to make this test: Let us suppose that the left eye is claimed to be defective. I grasp the patient's head with my left hand, ostensibly to steady it, then drop my thumb down in front of his right eye and ask him to read the test-type. This he does before he realizes what I am doing. Then I cover also his left eye with my right hand and he is unable to read at all.

Dr. Edward Jackson, Denver: My chief purpose was to urge that each of us should cultivate the ability to make an objective diagnosis of conditions presented and the probable extent of blindness that they cause, and my apology for introducing most of the several hundred cases is to indicate how, in going over my own cases, where I can interpret the meaning of the symptoms, I have been able to reach certain conclusions for myself. While comparison with cases of others is a very important thing, after that has been attended to each of us must get such experience with the ophthalmoscope and such acquaintance with conditions presented by the ophthalmoscope, in patients whose full cooperation we are sure of. When we get a sort of scale of objective conditions, coordinating with different degrees of blindness, we will be in a position where we can speak with as much definiteness as surgeons speak of the roentgen-ray findings, or of the presence of abscess, or a fracture, or as the psychiatrists speak as to the certainty or uncertainty of the cases on which they are expected to give expert opinions. The ophthalmologist should be trained to give an expert opinion of equal weight and accuracy with those based on roentgen-ray findings in many cases that are now arising in which blindness, partial or complete, is claimed. Even if we depend, to some extent on the subjective tests, we have the objective evidence and these objective tests should stimulate us to repeated subjective tests when we feel the results of the first obtained are not accurate.

PARENCHYMAL RÂLES IN PULMONARY TUBERCULOSIS

THEIR CLINICAL SIGNIFICANCE *

E. H. BRUNS, M.D.
Lieutenant Colonel, Medical Corps, United States Army
DENVER

It is not my intention in this paper to present another classification of râles, nor is it proposed to attempt to describe any new adventitious sounds elicited in pulmonary tuberculosis. During my rather long experience in tuberculosis work, râles have always presented the most perplexing signs in the physical examination of the lungs, especially when it came to correlating certain types of râles and the explanation of their production, as commonly taught, with other clinical findings. The standard books on physical diagnosis seem to have taken for granted the correctness of the older authors' conception of râles, and little that is new and more enlightening has been written recently on this subject. Writers have from time to time called attention to the fact that râles may persist for a long time after the patient has recovered. Amberson,[1] in one of his late articles, mentions such cases in which, with persisting râles, clinical symptoms have subsided, and serial roentgenograms have shown a very definite clearing. A certain group or class of râles, usually elicited only by expiratory cough and inspiration, is generally accepted as an important sign of chronic parenchymatous tuberculous lesions, and it is generally believed and held that these râles are produced by moisture or secretions in the terminal bronchioles. My object in this paper, based on careful study and investigations, is to show that this is probably not the case; that some other mechanism is responsible for their production; that their production seems to depend on a certain degree of collapse of the secondary lung lobules; furthermore, that these râles, while usually regarded as typical of tuberculosis, may be present without active lesions, and therefore they cannot be accepted as conclusive evidence of active or even manifest tuberculosis unless supported by other findings.

CLASSIFICATION

The nomenclature of râles, founded on many different classifications, makes it rather difficult to discuss a particular type, so that those who do not use the same terminology and classification will understand clearly the variety of râles being considered. Râles are classified according to their character, their method of production, their origin, or a combination of these. They may originate in the trachea, bronchial tree, cavities and pleura, but the discussion in this paper will pertain more to adventitious sounds coming from the pulmonary parenchyma. In considering the râles described by various authors, it may assist in the identification of parenchymal râles if those are referred to which can probably be classified in this group. Bushnell[2] introduced the term "indeterminate" râles to include râles which accompany inflammatory or irritative conditions of a less acuity than that of tuberculous pneumonia. He says, "Just as the crepitant and subcrepitant râle is associated with pneumonia, the indeterminate râle is the characteristic râle of chronic pulmonary tuberculosis.[3] He further subdivided indeterminate râles into "typical" and "atypical"; the typical, either small or medium, being uniform in size and occurring in showers, "the atypical found in tubes which contain varying and usually relatively small amounts of fluid, having no fixed characteristics as respects size, and time of occurrence in respiration."

---

* From the Fitzsimons General Hospital.

[1] Amberson, J. B., Jr.: Clinical Studies of the Healing of Pulmonary Tuberculosis, Am. Rev. Tuberc. **10:** 227 (Nov.) 1924.

Other authors (Flint[4]) employed the same term, applying it more specifically to a group of pulmonary râles elicited in tuberculosis of the lungs and other pulmonary conditions, the exact source and method of production of which are unknown. The râles generally encountered in pulmonary tuberculosis are the râles described by Bushnell and named "indeterminate." They were considered by him to be bronchial in origin, resulting from secretions in the bronchial tubes. This explanation for the "typical" form can be questioned for many reasons to be discussed later; but, granted that such is the case, even though their association with active disease could then be readily understood, this interpretation might still be misleading. Bushnell further says:

> Râles are the best evidence that the lesion is resisting its foes. Râles are absent in the obsolete or arrested lesion—the body does not need to fight. They are present in the stage of reaction—the body is fighting, whether successfully or not is to be determined in part by the number and quality of the râles, in part by other considerations. . . . They may be absent again, when the body can no longer fight—when the power to react is lost. Nothing could be more erroneous than to draw favorable conclusions from the diminution or the disappearance of râles in the very advanced case.

Turban[5] speaks of fine crepitations which, he says,

> probably originate from separation of alveolar walls which have become glued together and may be recognized in simple atelectasis. . . . The interpretation given of crepitation appears to me to be applicable in certain cases to somewhat coarser sounds. In cases running a favorable course, for years after cough and expectoration has disappeared, there may often be heard the same fine and medium râles which were to be heard at the height of the disease, especially after an effort to cough. It is impossible to distinguish these sounds from the true râles dependent on catarrh.

Sahli[6] refers to moist or bubbling râles, crackling dry râles and crepitations, all of which, according to his description, might be included under parenchymal râles. He describes the production of bubbling râles and crackling râles as "due to the formation of membranes of secretion in the bronchial lumen, which is stretched and torn apart again, partly by the movement of the air and partly by the movement of the lungs." The crepitations he conceives as being due to "the tearing apart of the alveolar walls by the inspiratory stream." He further adds that they can be produced by "blowing up the absolutely dry lung of the cadaver; and in perfectly healthy individuals with no real secretion in the alveoli or bronchi, crepitations are sometimes heard after they have been breathing superficially for a long time and then take a deep breath."

INTERPRETATION OF RÂLES

If we hope to interpret râles properly and use them as a practical guide in determining the character of a lesion and the clinical progress of a case, we must have a more accurate knowledge of their origin and the mechanism of their production. To have the term "bronchial" or "bubbling" apply to râles in cases in which, owing to the nature of the disease, we can assume them to be due to secretions in the bronchial tubes, and the term "indeterminate" to the same râles when the rationale of their production and the pathologic condition which they represent is uncertain, at least suggests that our interpretation in the former was erroneous.

In this paper I shall discuss a type of râles, small or medium, but uniform in size, bubbling, crackling, or crumpling in character, elicited toward the end of forced inspiration or bursting with the expiratory cough, and continuing during the greater part of the following inspiration. This is the common type of râle elicited over tuberculous lesions and designated at times as indeterminate, crepitations, cracklings, or fine and medium râles. We know that the sonorous and sibilant râles sometimes classified as whistling râles,[7] the bubbling râles of pulmonary edema and the gurgling râles of bronchiectasis are bronchial râles, but their law of production is different from that of the râles just described, which Bushnell would probably have designated as "typical indeterminate" râles. The former are variable, depending on the character and amount of secretions in the bronchial tubes or the turgescence of the mucous membrane; they may be heard at any time during respiration, and a cough is not required to elicit them. Sometimes a cough causes them to disappear, or they vanish after the patient raises some sputum.

From their sound, the method by which they are elicited and their behavior, no skepticism can exist as to their origin. In marked contrast, the others are localized and constant, regardless of the quantity of the patient's expectoration. They remain unchanged, irrespective of how much the patient coughs or raises during the examination. A cough is often necessary in order to elicit them. They may be fine or medium, but they are uniform in size and occur in showers at the same phase of respiration. They are encountered in all the pathologic-anatomic types of tuberculosis. The lesions may caseate and progress; they may fibrose and heal; perifocal reactions may develop and subside; pleural adhesions may be present or absent; the patient may have severe constitutional symptoms or none at all; he may have considerable cough with profuse expectoration or no cough and no expectoration; the roentgen ray may show any type of tuberculosis, and the involvement may be dense or it may be difficult to detect; still these râles remain unchanged and can persist indefinitely.

It is true that not infrequently, in time, as the lesions clear up and heal, the râles disappear, but we see many cases in which they persist after the patient has become cured and the roentgen ray reveals fibrous tuberculosis. It is inconceivable that secretions of invariable consistency and of such constancy could exist in the bronchial tubes, producing crackles and bubbles of uni-

---

[2] Bushnell, G. E.: Manifest Pulmonary Tuberculosis, Mil. Surgeon **42**: 383 (April) 1918.

[3] Bushnell, G. E.: The Diagnosis of Tuberculosis in the Military Service, Mil. Surgeon **40**: 620 (June) 1917.

[4] Emerson: Flint's Physical Diagnosis.

[5] Turban: The Diagnosis of Tuberculosis of the Lung.

[6] Potter: Sahli's Diagnostic Methods.

[7] Kaufman, I.: A Method of Teaching Students the Diagnosis of Pulmonary Tuberculosis, Am. Rev. Tuberc. **10**: 210 (Oct.) 1924.

Bro-Dart
INDUSTRIES
· NEWARK 5, N. J.
· LOS ANGELES 25, CALIF.
· TORONTO 6, CANADA
Made in U.S.A.